USAATCOM Technical Report 92-A-001

1N-52 159305 p.28

Autonomic Physiological Data Associated with Simulator Discomfort

James C. Miller, Thomas J. Sharkey, Glenna A. Graham, and Michael E. McCauley

(NASA-CR-177609) AUTONOMIC PHYSIOLOGICAL DATA ASSOCIATED WITH SIMULATOR DISCOMFORT (Monterey Technologies) 28 p

N93-24738

Unclas

G3/52 0159305

CONTRACT NAS2-12927 February 1993





ar = v v

Autonomic Physiological Data Associated with Simulator Discomfort

James C. Miller, Thomas J. Sharkey, Glenna A. Graham, and Michael E. McCauley

Monterey Technologies, Inc. P. O. Box 223699 Carmel, CA 93922

Prepared for Ames Research Center CONTRACT NAS2-12927 February 1993



Ames Research Center Moffett Field, California 94035-1000



			•
			•
			•
			•
-			

.

AUTONOMIC PHYSIOLOGICAL DATA ASSOCIATED WITH SIMULATOR DISCOMFORT

INTRODUCTION

There has existed a controversy concerning the relationships of autonomic functions to motion sickness for at least two decades. Money exhaustively reviewed the signs and symptoms of motion sickness (1). For those organs innervated by the autonomic nervous system (ANS), he noted that conflicting observations (e.g., both increases and decreases in heart rate) had been reported.

Money argued (1), in part, that since (a) visual-vestibular interactions reside in the central nervous system (CNS), (b) emesis has strong somatic muscular components (diaphragm and abdominal wall), (c) skin pallor can be induced at the sympathetic ganglion by acetylcholinesterase, and (d) that nausea may reflect reactions to CNS events alone, then one may not assume that motion sickness is a dysfunction within the autonomic nervous system (ANS). An alternative hypothesis is that motion sickness is a CNS dysfunction which usually has sequelae involving the ANS.

Subsequently, Graybiel and Lackner were unable to find systematic autonomic indicators of motion sickness within or across subjects (2). The measures available to the investigators were somewhat non-specific: heart rate, blood pressure, and body temperature are all subject to many intrinsic and extrinsic influences. However, Cowings et al. found systematic autonomic responses, then suggested that stability of ANS responding exists within subjects (3, 4). They used somewhat more specific measures than Graybiel and Lackner, including skin conductance and finger pulse volume, among others.

In the meantime, Stern and his colleagues at Pennsylvania State University showed across-subject consistency in the hypergastric response to motion, and anecdotal evidence of a correlation between hypergastria and subjective reports of gastric discomfort and nausea (5, 6, 7).

This somewhat confused state of affairs concerning ANS responses to motion sickness presented a problem to applied researchers associated with the U.S. Army's fixed-base, virtual-world helicopter simulator at NASA Ames Research Center. In the realm of engineering, the device provided a providential proving ground for controls and displays research. Unfortunately, a simulator sickness rate of 20 to 40% was expected for experienced pilots operating the device (8).

We report here the development of a physiological monitoring capability for the Army's Crew Station Research and Development Facility (CSRDF) and some preliminary physiological data. The objective this effort was to demonstrate sensitivity of the physiological measures in this simulator to self-reported simulator sickness.

METHOD

Eight channels of data were gathered through a 12-bit (4096-step) analog-to-digital system (Quatec, Akron OH, Model SAC-12) with a total sampling rate of 33 mHz. Six channels were autonomic physiological data: electrocardiogram (ECG), forearm skin conductance level (SCL), electrogastrogram (EGG), chest circumference for ventilatory rate (f_v), fingertip pulse (BVP), and fingertip skin temperature (T_{sk}). One channel was an event marker for flight segment time registration. The last channel was unused.

Signal Acquisition

The six physiological signal conditioners, built by NASA-Ames, were isolated at the power supply (voltage converter) level. They used front-end band-pass filtering specific to the signal of interest, and amplified each signal to the ±5 v p-p range. The differential amplifiers used for ECG and EGG had common mode rejection ratios exceeding 80 db.

The adhesive spot electrodes used for ECG, EGG, and SCL were of a standard Ag/AgCl clinical ECG variety with snap connectors (Vermont Medical, Inc., Bellows Falls, VT 05101, Re-order no. A-10005). These electrodes were recessed, with a thin piece of foam between the skin and the electrode surface, to minimize motion artifact. Clinical electrolyte (TECA Corp., Pleasantville, NY 10570, Cat. no. 822-201210, NDC 0324-0045-06) was used at the electrode-skin interface.

Creation of, and access to, the digitized data files was mediated by the software, CODAS (Data-Q Instruments, Inc., Akron OH, version 3.0), which ran at a total sampling rate of 800 Hz (100 Hz per channel). CODAS also supported visual data display and ASCII or Lotus 123 file creation.

Electrogastrogram - The EGG signal conditioner analog band pass, 0.02 to 0.25 Hz (1.2 to 15 cpm), allowed the detection of "hypergastria" in the 4 to 9 cpm range (6). We used the two active EGG electrode placements of Stern et al. (6): (1) 10 cm above the navel and 6 cm left of the centerline, and (2) 4 cm above the navel on the centerline, with a reference electrode placed on the left mid-axillary line, halfway between the iliac crest of the hip and the bottom of the rib cage.

Skin Conductance Level - Though the hypothalamic control of eccrine thermoregulatory sweating is well described, and the responsiveness of the SCL, mediated by eccrine sweat gland activity, to motion discomfort has been noted (3), the complete

picture of the neural circuitry underlying eccrine gland activity is not clear (9). However, decades of experience suggest that the SCL may provide reasonable indications of emotion-triggered activity in the sympathetic branch of the ANS.

Due to the requirement that the pilot use his hands to fly, and the impracticality of electrode placements on the sole of the foot, the two SCL electrodes were attached to the skin of the medial wrist. They were placed laterally to one another, just medial to the anterior annular ligament. The skin was not abraded.

The SCL signal conditioner placed a 15 Hz, constant 1.25 v p-p square wave across the skin and a 100 kohm current limiting resistor in series with the skin. SCL variations were viewed in the frequency range dc to 2 hz as the voltage required to hold current constant across the combined resistive impedances. The current flow through the series resistances varied as allowed by skin conductance, the inverse of resistance. The signal conditioner voltage output was nonlinear, and was corrected by the formula,

$$r_{skin} = \frac{gain}{voltage-offset} - 100 K\Omega$$

where

gain = 1,453,682 and
offset = -5.73354 volts

The SCL, in mhos (a unit of electrical conductance), was calculated as the reciprocal of $r_{\rm skin}$. Conductance has been preferred to resistance in electrodermal response analysis, due to a more Gaussian distribution, for a number of years (10).

Electrocardiogram and Vagal Tone - The ECG provides a window into electrical conduction characteristics of the cardiac neuromuscular structure. It is also one technique used for the measurement of heart rate. Both conduction and rate are modified

by autonomic humoral mechanisms, and rate is modified by intrinsic myocardial length-tension relationships (the Frank-Starling mechanism).

In addition, rate is influenced by autonomic parasympathetic fibers travelling in the tenth cranial nerve, the vagus. Efferent cardiac fibers originate in the medulla and travel in the vagus. Also, the efferent arms of reflex circuits originating from pulmonary and cardiac receptors travel in the vagus. Rate is also influenced by autonomic sympathetic fibers originating in the cardiac nuclei of the sympathetic trunk.

The reflex effects of ventilatory rate and depth on heart rate are well documented (11). Scientific literature published over 70 years ago showed "(1) that respiratory sinus arrhythmia is mediated by the vagus; (2) that the amplitude of respiratory sinus arrhythmia is related to the functional status of the cardiac vagi (i.e., cardiac vagal tone); and (3) that an individual with pronounced respiratory sinus arrhythmia has specific behavioral characteristics." (12)

Investigators have long sought a window on vagal activity by quantifying respiratory sinus arrhythmia. Recently, several laboratories independently identified three bandwidths of heart rate variation (13). In humans, studies have indicated relationships between heart rate variations around 0.10 Hz and mental work load (14, 15). The responses of the .12 to 4.0 Hz component of sinus arrhythmia to atropine (16) and in the presence of alpha- and beta-adrenoreceptor blockers indicates its mediation by the parasympathetic branch of the autonomic nervous system, in particular, the vagus nerve within which the applicable fibers run (17).

The Vagal Tone Monitor (VTM; Delta-Biometrics, Bethesda, MD) provided automated, on-line data reduction from the raw ECG to one component of respiratory sinus arrhythmia. We used the VTM to assess vagal activity, or vagal tone (VT), monitoring the middle component of sinus arrhythmia, the 0.12 to 0.40 Hz band. This provided us with an index of parasympathetic activity. The

VTM also provided a cardiotachometer function from which we extracted the directly-measured heart period (HP), the reciprocal of heart rate. HP was selected as a measure, rather than heart rate, for its more Gaussian distribution.

The ECG signal was derived from electrodes placed to generate the "CR-5" lead (18). The CR-5 lead is an electrode placement which minimizes movement and EMG artifacts in the ECG and produces a lead-II-appearing (large R wave) tracing. The lead was described by Blackburn, who investigated the characteristics of 22 different bipolar chest leads (19). The "C5" lead was recorded from the manubrium of the sternum or from the right clavicle to the C5 position (left anterior axillary line at the 5th intercostal space. It was the most sensitive lead for the detection of S-T segment changes after exercise. We used the standard electrocardiography color-code and nomenclature for electrode connectors. The white lead (RA), to minimize motion and EMG artifact from the clavicular portion of the pectoralis major, was placed when possible upon the manubrium of the sternum, just below the notch at the top of the sternum. pectoral activity in this experiment was minimal, positions at the level of the manubrium, but displaced to the subject's right, were used when chest hair patterns were a problem.

The black lead (LA) was attached directly below the left nipple in the 5th intercostal space. We used finger pressure on the rib cage to find the intercostal space. Placement directly over a rib might have caused motion artifact. The RA and LA connections led to the active inputs of a differential amplifier with analog front-end band-pass filtering at 0.5 to 100 Hz. The green (RA; reference electrode) lead was attached slightly forward of the right mid-axillary line, halfway between the iliac crest of the hip and the bottom of the rib cage. Placement on the iliac crest or on the lower ribs might have caused motion artifact.

Subjective and Other Evaluations

The pilots were asked to rate their motion discomfort (MD) on a 7-point scale, on which 1 was "Normal, symptom free," 7 was "Severe discomfort, I am unable to continue," and the values 2 through 6 were not anchored (8). The ratings were collected once every five minutes while the pilots were in the simulator. Also, the pilots completed a motion history questionnaire and a simulator side effects questionnaire (SSEQ). In addition, they received two postural equilibrium assessments: a stand-on-one-leg, eyes-closed test (SOLEC) and a walk-on-straight-line, eyes-closed test (WOFEC). The measures of interest in the latter were the total time of balance across three trials (SOLEC) and the total number of heel-to-toe steps accomplished prior to loss of balance in one trial (WOFEC).

Data Reduction

All channels of analog physiological data were sampled at 100 samples/sec, then reduced in the following manner. The skin conductance level (SCL) data were summarized as mean values for 30-second epochs. Then the linear trend was calculated, using the least squares method, for each session, and subtracted from the raw data. The residual 30-second epoch means were used for further analyses. These 30-second means fell into the domain of tonic, rather than phasic, SCL measures.

Cardiac interbeat intervals, or heart periods (HP), were also summarized as mean values for 30-second epochs. The variance in cardiac interbeat interval was partitioned such that variance in the frequency band, 0.12 to 0.40 Hz, was reported each 30 seconds as vagal tone (VT). The VT data were smoothed by averaging together four sequential epochs (two minutes), creating a moving

average lagged 30 seconds at a time and labelled by the fourth epoch in the average.

For subsequent analyses involving the EGG data, the HP, SCL, and VT data were reduced to 1-minute epochs by averaging together sequential pairs of 30-second epochs, without overlap.

The digitized EGG data for one minute (6000 samples) were shifted to zero mean, tapered at both ends (10% taper) (20), and subjected to a discrete Fourier transform (MATLAB, The Math Works, Inc., South Natick, MA). The output of the transform included raw energy estimates and phase estimates in each frequency bin from 1 through 13 cycles/minute. The energy estimates were reduced to an index of normal gastroenteric activity (EGG3; 3 cycles/min) and of hypergastria (HG; mean of 4 through 9 cycles/min) (5). The raw power data were transformed to logarithms to provide more Gaussian data distributions. The two indices were smoothed by averaging together four sequential minutes of log power data, creating a moving average lagged one minute at a time and labelled by the fourth minute in the average.

The MD ratings were generalized across 5-minute periods. Each rating value was assigned to the minute in which it was reported, and to the two minutes immediately preceding and immediately following the report. For example, the report "3" in minute 20 and "4" in minute 25 caused minutes 18, 19, 20, 21, and 22 to be assigned the value "3," and minutes 23, 24, 25, 26, and 27 to be assigned the MD rating "4." When a 2-rank change occurred, the rating value was assigned to the minute in which it was reported, and the one minute immediately preceding or immediately following the report, depending on the sequence of change. The other, more distant minute preceding or following the report was assigned the next report level. For example, the report "2" in minute 20 and "4" in minute 25 caused the sequence 2-2-2-2-3-4-4-4-4, for minutes 18 through 27, respectively. No 3-rank changes occurred across sequential 5-min periods.

Flight Conditions

The pilots flew in four experimental conditions, created from two experimental factors with two levels each. The conditions were low-effort maneuvering at 100 ft above ground level (AGL), low-effort maneuvering at 400 ft AGL, high-effort maneuvering at 100 ft AGL, and high-effort maneuvering at 400 ft AGL. The flight conditions, pilot performance data, equilibrium data, and motion discomfort data are reported fully elsewhere (21). The full analysis indicated that only the altitude factor had a significant effect upon motion discomfort. Thus, a subset of data were selected for an analysis here of physiological data which would reflect the effects of the altitude manipulation. The data came from two independent groups of subjects with n=6 at 100 ft AGL and n=7 at 400 ft AGL.

Statistical Analyses

Graphic displays of the individual and group physiological data were used to search for apparent differences related to altitudes and related to motion discomfort ratings. For the group data, when a MD contrast was desired, an approximate median split approach was used. The subjects were separated into a low ratings group (maximum ratings of "1" through "3") and a high ratings group (maximum ratings of "4" through "7").

The data set was not complete enough nor extensive enough to attempt to partition variance among MD, subjects and error factors using an analysis of variance. However, the data contributing to the graphic views of the data allowed a simplistic sensitivity assessment of the different physiological measures. The metric used for sensitivity assessment was the ratio of the average, between-condition difference (d) and the

grand standard deviation (s). The latter was calculated from all available observations of the variable.

Discriminant analyses (StatGraphics, STSC, Inc., Rockville MD, version 4.0) were used to search for predictive relationships between motion discomfort ratings and a multivariate set of physiological activities. This approach was used for individual and for group data. For discriminant analyses using group data, 5-minute MD ratings were categorized as low (ratings of "1"), medium (ratings of "2" and "3"), and high (ratings of "4" through "7"). For discriminant analyses using individual data, the 5minute MD ratings were not combined. Five physiological variables were used to predict MD: HP, SCL, HG, EGG3 and VT. Each variable was represented as within-subject, within-session, within-variable standard scores (mean=0, s=1). The variable, time, was purposely avoided as a predictor to allow a full exploration of the predictability of MD from these physiological data. Not enough data were available to both "train" and validate the discriminant functions within or across subjects.

RESULTS

We recovered usable SCL data from 11 subjects, HP and VT data from 13 subjects, EGG data from 11 subjects, and MD ratings from 9 subjects, as shown in Table 1. Eight subjects provided complete sets of data.

Standard score data for the five physiological variables, compiled across the eight subjects with complete data sets, were relatively Gaussian for each variable (Figure 1).

TABLE 1. DATA RECOVERY, BY SUBJECTS AND VARIABLES, AND MAXIMUM MOTION DISCOMFORT (MD) RATINGS. SCL, SKIN CONDUCTANCE LEVEL; HP, HEART PERIOD; VT, VAGAL TONE; EGG, ELECTROGASTROGRAM; ALL, ALL DATA AVAILABLE.

Subj.	Alt	SCL	HP & VT	EGG	MD	Max MD	All
S02B	HIGH	х	х	х	Х	1	х
S03A	HIGH	x	х	Х	Х	4	Х
S04A	LOW	х	Х	Х	Х	2	Х
S05A	LOW	х	х				
S06A	HIGH	х	х	Х			
S07A	LOW		x				
S08A	HIGH	Х	x	Х	Х	6	Х
S09A	LOW	х	Х	х	х	5	х
S10A	HIGH		х	Х	x	2	
S12A	HIGH	х	х	х	x	3	х
S14A	HIGH	х	х	х	х	3	х
S15A	LOW	х	х	х			
S16A	LOW	х	х	х	х	4	х

Graphic Views

The group mean HP data revealed a systematic difference for the altitude treatment (Figure 2). The heart rate of the low-altitude group (n=6) was consistently higher (shorter HP) than the heart rate of the high-altitude group (n=7). The mean HP difference between the low and high altitude conditions, for epochs 1 through 60, was 84.4 msec. The grand standard deviation of all observations (1522 observations) in the supporting data set was 88.5 msec. The ratio, d/s, was .95.

The group mean HP data also revealed a systematic difference for the MD median split (Figure 3). The heart rate of the low-rating group (n=5) was consistently lower (longer HP) than the heart rate of the high-rating group (n=4). The mean d for HP between

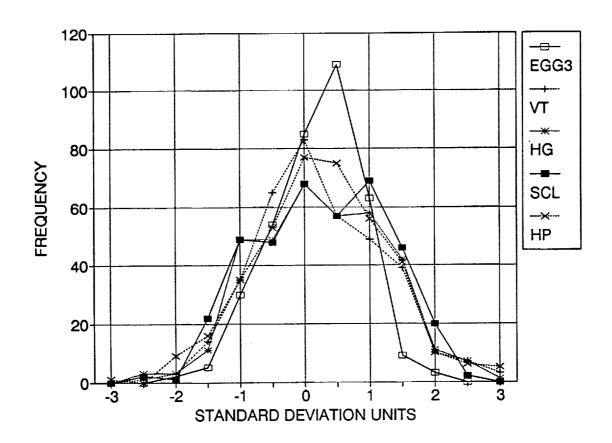


Figure 1.- Frequency distributions of across-subject observations, using within-subject standard scores, of normal gastric activity (EGG3; log power), vagal tone (VT), hypergastria (HG; log power), tonic skin conductance level (SCL; mhos), and heart period (HP; milliseconds). Numbers of subjects and observations available in Table 2.

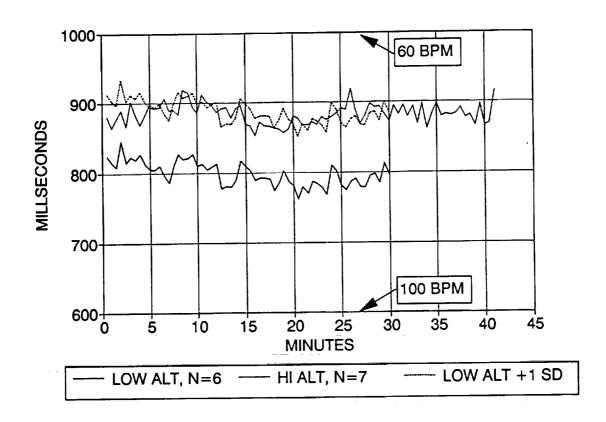


Figure 2.- Mean heart period data from low (100 ft AGL) and high (400 ft AGL) altitude groups, and one grand standard deviation (Table 2) above the low altitude mean.

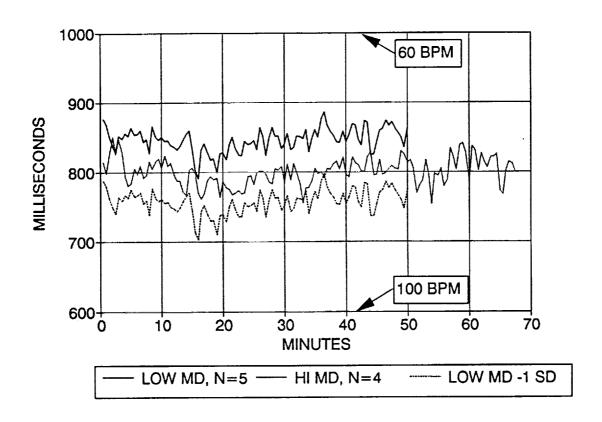


Figure 3.- Mean heart period data from low (1-3) and high (4-7) motion discomfort (MD) rating groups, and one grand standard deviation (Table 2) below the low MD mean.

low and high MD categories, for epochs 1 through 100, was 47.6 msec, and the ratio, d/s, was .54.

The group mean SCL data revealed no systematic differences for the altitude treatment or for the MD median split. The group mean VT data revealed a systematic difference for the altitude treatment (Figure 4), but none for the MD median split. The VT of the low-altitude group (n=6) was consistently less than the VT of the high-altitude group (n=7). The mean plot of the low-rating subjects overlaid that of the high-rating subjects. The mean d for VT between low and high altitudes, for epochs 1 through 60, was .86 units. The grand VT s (1482 observations) was .98 units, and the ratio, d/s, was .88.

The group mean EGG3 data revealed no systematic differences for the altitude or MD comparisons. The group mean HG data revealed a systematic difference for the altitude treatment (Figure 5). The HG of the low-altitude group (n=4) increased steadily through minute 29, while the HG of the high altitude group (n=7) held steady. For minutes 5 through 10 plus minutes 25 through 30, the HG mean log power d was .57, the grand s (524 observations) was .84, and the ratio, d/s, was .68.

The group mean HG data also revealed a systematic difference for the MD median split (Figure 6). The HG of the low-rating group (n=5) diminished, while the HG of the high-rating group (n=4) increased. For minutes 30 through 50, the HG mean log power, d, was .84, and the ratio, d/s, was 1.0.

These calculations are summarized in Table 2. The rank order of sensitivity was:

- 1. HG for MD (d/s=1.0)
- 2. HP for altitude (.98)
- 3. VT for altitude (.88)
- 4. HG for altitude (.68)
- 5. HP for MD (.54)

Discriminant Analyses

Individual discriminant analyses, performed on the data of each of the six subjects with maximum MD ratings greater than "2," revealed an idiosyncratic predictability of MD ratings. The six analyses produced solutions ranging from two to five functions. The first functions explained 64 to 93% (median=82.5%), and the second functions explained 4.5 to 38% (median=14.5%), of the variance in the subjects' data. All first functions were highly

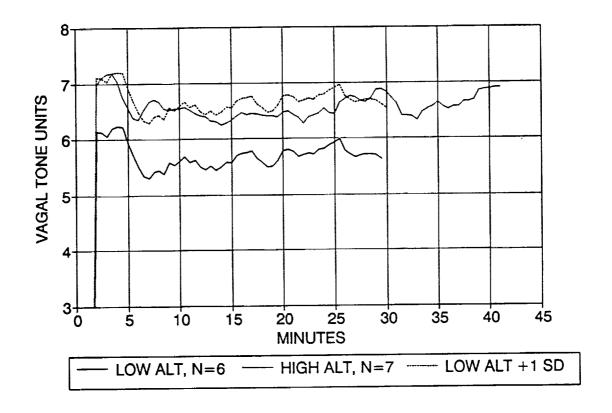


Figure 4.- Mean vagal tone data from low (100 ft AGL) and high (400 ft AGL) altitude groups, and one grand standard deviation (Table 2) above the low altitude mean.

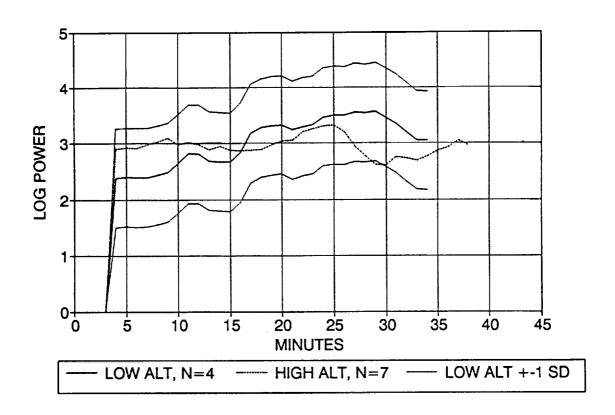


Figure 5.- Mean hypergastria data from low (100 ft AGL) and high (400 ft AGL) altitude groups, and one grand standard deviation (Table 2) above the low altitude mean (minutes 5-10) and below the low altitude mean (minutes 25-30).

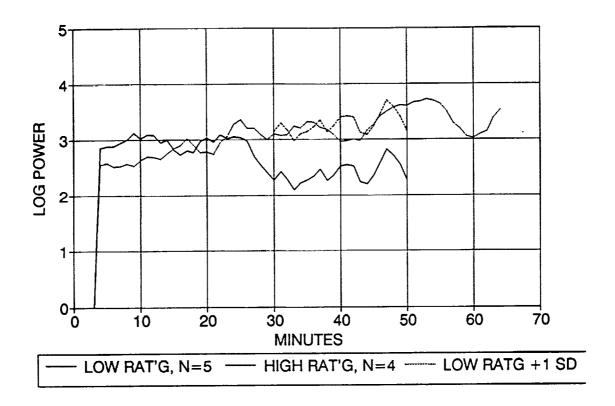


Figure 6.- Mean hypergastria data from low (1-3) and high (4-7) motion discomfort (MD) rating groups, and one grand standard deviation (Table 2) above the low MD mean (minutes 30-50).

TABLE 2. SUMMARY OF MEAN DIFFERENCE (d) AND GRAND STANDARD DEVIATION (s) DATA FOR FIVE PHYSIOLOGICAL MEASURES IN ALTITUDE (100 vs. 400 ft AGL) AND MOTION DISCOMFORT (1-3 vs. 4-7) COMPARISONS.

	EGG	HG	HP	SCL	VT			
	log power		msec	mhos	units			
Grand s	1.12	.84	88.5	2.40x10- 6	.98			
n	11	11	13	11	13			
No. of Observations	505	524	1522	1217	1482			
Motion Discomf	Motion Discomfort:							
d	-	.84	47.6					
d/s		1.0	.54					
Altitude:								
đ	-	.57	84.4		.86			
d/s	-	.68	.95		.88			

significant (p<.001) by Chi². Three second functions were significant (p<.05).

No single physiological variable dominated the top of the order completely (Table 3). The Friedman two-way analysis of variance (22), applied to the column-sums of Table 3, produced a moderately significant result (Chi, = 7.07, df=4, p<.20). SCL entered first into the first function three times, and second once. HG entered first into the first function once, and second twice.

However, these individual discriminant analyses provided better than expected (50%) correct classifications of within-subject MD reports (Table 4). The correct classifications ranged from 80% to 100% across the MD categories.

These data and the data of two more subjects, <u>combined</u> across subjects as within-subject, within-session, within-variable standard scores (mean=0, s=1), produced correct classifications of near or less than 50% across MD groups. This analysis was based upon the eight subjects with complete data sets (Table 1) and the grouping of MD ratings into three categories, low, medium, and high. The correct classifications for these groups

were 38% (62 minutes), 29% (39 minutes), and 54% (33 minutes), respectively.

TABLE 3. RANK ORDERS OF FIRST-FUNCTION, STANDARDIZED COEFFICIENTS FROM DISCRIMINANT ANALYSES FOR SIX SUBJECTS, PREDICTING MOTION DISCOMFORT (MD) FROM NORMAL GASTROENTERIC ACTIVITY (EGG3), HYPERGASTRIA (HG), HEART PERIOD (HP), SKIN CONDUCTANCE LEVEL (SCL), AND VAGAL TONE (VT).

Subject	EGG3	HG	HP	SCL	TV
S12	5	2	3	1	4
S14	2	3	1	4	· 5
S16	5	4	3	1	2
S3	3	2	4	1	5
S8	3	1	5	2	4
S9	1	2	4	3	5
Sums	19	14	20	12	25

An examination of within-subject intercorrelation matrices revealed no systematic pattern. First, we expected to find a relationship between VT and HP. However, the Pearson r values were small and variable, ranging across subjects from -.029 to .420 (mean, via Fisher z = .122, df from 37 to 59). Second, we expected to find a relationship between VT and HG. However, the Pearson r values ranged from -.429 to .442. Generally, HG and EGG3 were highly negatively correlated.

CONCLUSIONS

The graphic views of the group mean data suggested some autonomic patterns. For the motion discomfort (MD) median split, higher MD ratings seemed to be associated with higher heart rates and higher HG activities (Figures 3 and 6). These responses to MD align with observations by Cowings et al. (3) and by Stern et al. (5).

TABLE 4. CORRECT CLASSIFICATIONS, FROM INDIVIDUAL DISCRIMINANT ANALYSES, BY SUBJECT (n=6) AND MOTION DISCOMFORT RATING (MD). NUMBER OF MINUTES CORRECTLY CLASSIFIED AND THE RESPECTIVE PERCENT CORRECT.

	MD Group					
Subject	1	2	3	4	5	6
S3	4	14	10	8		
*	100	93	91	89		
S8	4	12	1	4	4	4
*	80	55	100	100	80	. 80
S9			4	20	5	
8			80	69	100	
S12	9	20	5			
૪	64	80	100			
S14	16	20	5			
8	100	100	100	1		
S16	26	4	1	3		
8	70	29	17	75		
n	5	5	6	4	2	1
Approximate Medians:						
# Min.	9	14	4.5	6	4.5	4
*	80	80	100	82	90	80

For the altitude comparison, heart period (HP) and vagal tone (VT) seemed to take on pilot mental workload connotations. Vagal tone was lower and heart rate was higher, suggesting higher workload at the lower altitude (100 ft AGL; Figures 2 and 4). This apparent workload effect was consistent with the views of pilots that flights at lower, nap-of-the-earth altitudes produce higher workloads than flights at higher altitudes.

However, the data supported an alternative hypothesis explaining the HP and VT differences at the two altitudes. The lower altitude seemed to produce a slight hypergastric disturbance (Figure 5), consistent with the hypothesis that a lower nap-of-the-earth altitude would produce such an effect, and the higher incidence of motion discomfort at the lower altitude (21). We cannot, from these data, determine whether workload or MD affected HP and VT.

The discriminant analyses suggested (1) that no single physiological variable dominates in the prediction of MD across subjects, (2) that physiological variables may predict MD when restricted to within-subject comparisons, and (3) that physiological variables may not predict MD when combined across subjects. There are four caveats. First, the capability to perform stepwise discriminant analyses would have allowed hypothesis testing when each physiological variable entered into a function (23), allowing a more definitive approach to the question of idiosyncrasies. This capability should be added to the analysis approach. Second, the small number of observations per individual subject (range 39 to 61) probably introduced sampling errors.

Third, we were unable to validate our discriminant functions within or across subjects, or with data from another investigation. This validation work is needed. Finally, the assumption that a single MD report may be generalized across several minutes obscured some unknown proportion of the true classifications of minutes. MD reporting once per minute should be attempted in the future.

The objective of this effort was to demonstrate the sensitivity of physiological measures in this simulator to self-reported simulator sickness. We were successful. However, we would hope to develop the metrics to somewhat greater levels of sensitivity in subsequent work. Figures 3, 5 and 6 and Tables 2 and 3 suggested that HP, HG, and SCL may be more sensitive to simulator sickness than the other two measures used here (VT and EGG3).

22

FOOTNOTES

¹Dr. Miller served as a consultant to Monterey Technologies. His firm is Miller Ergonomics, Lakeside, California.

ACKNOWLEDGEMENTS

A version of this material was presented at the 4th annual NASA Simulator Steering Committee meeting, Orlando, Florida, 12-13 March 1991.

The project was supported by the U.S. Army Aeroflight Dynamics Directorate, contract NAS2-12927, initiated by Maj. James W. Voorhees, Ph.D. and monitored by K. Perlaki. The excellent technical support by Scott Jensen of NASA/Ames Research Center is gratefully acknowledged.

The opinions expressed in this report are those of the authors and do not necessarily reflect the official view of the Army or of NASA; and they do not constitute a standard, specification or regulation.

REFERENCES

- 1. Money KE. Motion Sickness. Physiol. Rev. 1970; 50:1-39.
- 2. Graybiel A, Lackner JR. Evaluation of the relationship between motion sickness symptomatology and blood pressure, heart rate, and body temperature. Aviation Space and Environmental Medicine. 1980; 51:211-214.
- 3. Cowings PS, Suter S, Toscano WB, Kamiya J, Naifeh K. General autonomic components of motion sickness. Psychophysiology. 1986; 23(5):542-551.
- 4. Cowings PS, Naifeh KH, Toscano WB. The stability of individual patterns of autonomic responses to motion sickness stimulation. Aviation Space and Environmental Medicine. 1990; 61:399-405.
- 5. Stern RM, Hu S, Anderson RB, Liebowitz HW, Koch KL. The effects of fixation and restricted visual field on vection-induced motion sickness. Aviation Space and Environmental Medicine. 1990; 61:712-715.
- 6. Stern RM, Hu S, Vasey MW, Koch KL. Adaptation to vection-induced symptoms of motion sickness. Aviation Space and Environmental Medicine. 1989; 60:566-572.
- 7. Stern RM, Koch KL, Stewart WR, Lindblad IM. Spectral analysis of tachygastria recorded during motion sickness. Gastroenterology. 1987; 92:92-97.
- 8. McCauley ME, Hettinger LJ, Sharkey TJ, Sinacori JB. The effects of simulator visual-motion asynchrony in simulator induced sickness. Paper presented at the AIAA Flight Simulation Technologies Conference, Dayton, OH, (Paper AIAA-90-3172). Sept 1990; p. 9.
- 9. Shields SA, MacDowell KA, Fairchild SB, Campbell ML. Is mediation of sweating cholinergic, adrenergic, or both? A comment on the literature. Psychophysiology. 1987; 24(3):312-319.
- 10. O'Donnell RD. Contributions of Psychophysiological Techniques to Aircraft Design and other Operational Problems (AGARD-AG-244). Neuilly sur Seine, France: NATO Advisory Group for Aerospace Research and Development, 1979.

Fi

- 11. Porges SW, McCabe PM, Yongue BG. Respiratory-heart interactions: psychophysiological implications for pathophysiology and behavior. In: Cacioppo JT, Petty RE, ed. Perspectives in Cardiovascular Psychophysiology. New York: The Guilford Press, 1982:223-264.
- 12. Akselrod S, Gordon D, Ubel FA, Shannon DC, Barger AC, Cohen RJ. Power spectrum analysis of heart rate fluctuation: a quantitative probe of beta-to-beat cardiovascular control. Science. 1981; 213:220-222.
- 13. Aasman J, Mulder G, Mulder LJM. Operator effort and the measurement of heart-rate variability. Human Factors. 1987; 29:161-170.
- 14. Vicente KJ, Thornton DC, Moray N. Spectral analysis of sinus arrhythmia: a measure of mental effort. Human Factors. 1987; 29:171-182.
- 15. Dellinger JA, Taylor HL, Porges SW. Atropine sulfate effects on aviator performance and on respiratory-heart period interactions. Aviation Space and Environmental Medicine. 1987; 58:333-338.
- 16. Grossman P, Stemmler G, Meinhardt E. Paced respiratory sinus arrhythmia as an index of cardiac parasympathetic tone during varying behavioral tasks. Psychophysiology. 1990; 27:404-415.
- 17. Simonson E. Physiology of Work Capacity and Fatigue. Springfield, IL: Charles C. Thomas, 1971.
- 18. H. Blackburn Ed. Measurement in Exercise Electrocardiography. 1967. Springfield, IL: Charles C. Thomas, 1969.
- 19. Bingham C, Godfrey MD, Tukey JW. Modern techniques of power spectrum estimation. IEEE Trans. Audio Electroacoust. 1967; AU-15:56-66.
- 20. McCauley ME, and Sharkey TJ. The effect of global visual flow on simulator sickness. Paper presented at the AIAA Flight Simulation Technologies Conference, New Orleans, LA, (Paper AIAA-91-2975-CP). August 1991; p. 9.
- 21. Siegel S. Non-Parametric Statistics for the Behavioral Sciences. New York: McGraw-Hill, 1956.

22. Cohen J, Cohen P. Applied Multiple Regression/Correlation Analysis for the Behavioral Sciences. New Jersey: Lawrence Erlbaum Associates, 1975.

REPORT DO	Form Approved OMB No. 0704-0188			
Public reporting burden for this collection of informa	aviewing instructions, searching existing data sources.			
gathering and maintaining the data needed, and con- collection of information, including suggestions for re Davis Highway, Suite 1204, Arlington, VA 22202-430	educing this hurden to Washington Hea	idouariers Services. Directorate to	arding this burden estimate or any other aspect of this or information Operations and Reports, 1215 Jefferson Project (0704-0188), Washington, DC 20503.	
1. AGENCY USE ONLY (Leave blank)	2. REPORT DATE		ND DATES COVERED	
	February 1993	Contractor Re	port	
4. TITLE AND SUBTITLE			5. FUNDING NUMBERS	
Autonomic Physiological Data	Associated with Simulat	or Discomfort	NIA 52 12027	
6. AUTHOR(S)			NAS2-12927	
James C. Miller, Thomas J. Sh.	arkey, Glenna A. Graham	ı, and	1	
Michael E. McCauley	•	•		
7. PERFORMING ORGANIZATION NAME	E/S) AND ADDRESS(ES)		8. PERFORMING ORGANIZATION	
		Dosaarch Center	REPORT NUMBER	
Monterey Technologies, Inc., (Moffett Field, CA 94035-1000				
U.S. Army Aviation and Troop	- ·		A-93026	
Moffett Field, CA 94035-1000		en contor,		
9. SPONSORING/MONITORING AGENCY		1	10. SPONSORING/MONITORING	
9. SPUNSUHING/MUNITURING AGENC	(NAME(3) AND ADDITECTION	,	AGENCY REPORT NUMBER	
National Aeronautics and Space	e Administration			
Washington, DC 20546-0001		and Troop	NASA CR-177609	
Command, St. Louis, MO 6312	20-1798		USAATCOM TR-92-A-001	
THE PARTY OF THE P				
11. SUPPLEMENTARY NOTES	1-1-: A Dasagrah Cont	NAC 2/12 / Moffett	E-14 CA 04025 1000-	
Point of Contact: Kinga M. Per (415) 604-500		er, IVIS 245-4, IVIOITEU	Field, CA 94055-1000,	
(413) 004-300	D.			
12a. DISTRIBUTION/AVAILABILITY STA	TEMENT		12b. DISTRIBUTION CODE	
Unclassified — Unlimited				
Subject Category 52				
13. ABSTRACT (Maximum 200 words)				
		4 141 . //		
We report here the develop	ment of a physiological m	onitoring capability to	or the Army's advanced helicopter	
simulator facility and some pr	reliminary physiological (data. Our objective w	vas to demonstrate sensitivity of	
physiological measures in this s	imulator to self-reported s	imulator sickness. 1118	e data suggested that heart period.	
• • •	ice level were more sensiu	ve to simulator sicknes	ss than were vagal tone and normal	
electrogastric activity.				
14. SUBJECT TERMS			15. NUMBER OF PAGES	
Simulator discomfort, Simulat				
Simulator sickness, Heart perio				
	SECURITY CLASSIFICATION	19. SECURITY CLASSIF	1100	
OF REPORT	OF THIS PAGE	OF ABSTRACT	10011011	

11

17. SECURITY CLASSIFICATION OF REPORT

18. SECURITY CLASSIFICATION OF THIS PAGE

Unclassified